THE ANOMALOUS ORIGIN OF THE LEFT CORONARY ARTERY FROM THE PULMONARY ARTERY

BY

JOHN D. KEITH

From the Department of Pædiatrics, Faculty of Medicine, University of Toronto, Canada

Received, June 3, 1958

Considerable variation in origin and distribution of the coronary arteries has been described over the years. One of the most interesting anomalies is that of the left coronary artery arising from the pulmonary artery in a heart without other defects, first described by Abrikosoff in 1911. Cases were reported sporadically in the next twenty years but clinical interest was not aroused until Bland, White, and Garland, in 1933, integrated the clinical and pathological data and recorded an electrocardiogram in an infant dying of this condition. In all, nearly 50 cases have been reported, and the author has had an opportunity of studying 10 more at the Hospital for Sick Children in recent years.

Bland *et al.* reported finding this aberrant left coronary once in 6800 necropsies at the Massachusetts General Hospital over a period of thirty-seven years. In the total group of congenital heart disease we have found this anomaly to occur in 0·5 per cent of cases. In relating it to child population in the Toronto Heart Registry it appears once in 300,000 children. It is undoubtedly a rare defect but its diagnosis during life is now possible in most cases, and successful surgery appears within reach.

Most instances of this defect have been found in babies who have died in the first year of life. However, survival into adult life is possible, and this appears to occur in approximately 15 per cent of cases. Kaunitz, in 1947, presented data on 7 such cases: the oldest was sixty-four and was first reported by Abbott in 1927. All of the adults were discovered, incidentally, at autopsy, and usually in subjects who had died suddenly without obvious clinical cause.

Pathology. At necropsy the origin of the left coronary artery is not immediately visible and attention is attracted first to the grossly enlarged left ventricle of aneurysmal proportions, with a ventricular wall that appears somewhat thin in proportion to its size. In most studies the left ventricular wall is actually normal in thickness and only occasionally is it either obviously hypertrophied or pathologically thin. However, as might be expected, the right ventricle by comparison is small and compressed. The total heart weight is increased (see Fig. 1). Patchy fibrosis is found in the left ventricle, particularly at the apex, and over the anterior portion of the myocardium (Fig. 2). It involves all layers down to the endocardium, and the latter shows a diffuse endocardial fibroelastosis. The fibrosis of the muscle of the left ventricle diminishes in degree as one approaches the collateral branches of the right coronary on either side. Histologically the muscle fibres are increased in number and may present a patchy disintegration. There is an increase in elastic tissue in the involved areas and often some œdema. In the more advanced cases calcification within the myocardium has been noted: Kaunitz (1947) reported this finding in slightly over half the cases.

Aided by a grant from the Ontario Heart Foundation, Toronto.

The lowered oxygen content of the blood in the pulmonary artery, which may or may not be available to the left coronary, is obviously not the cause of the pathological changes in the muscle of the left ventricle, since severely cyanotic children with various other types of congenital heart disease do not show such histological changes.

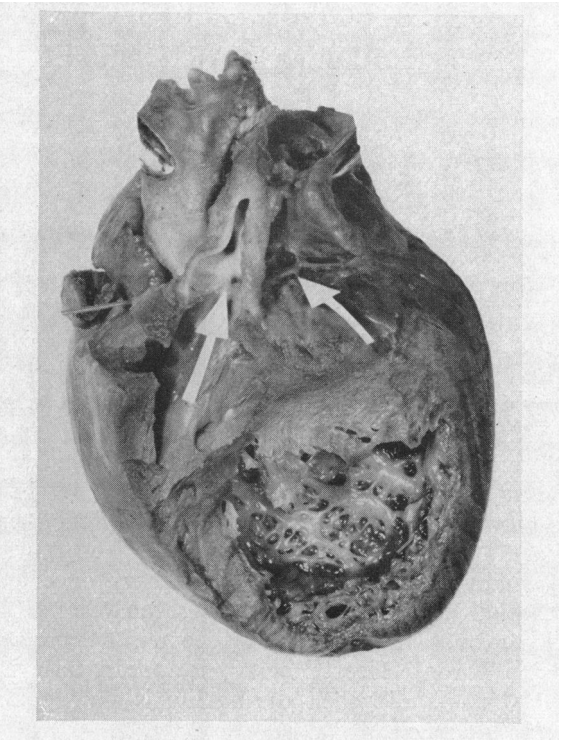

FIG. 1.—Arrows show orign and early course of left coronary artery. See also large left ventricle and small right ventricle.

The right coronary arises from its usual position, and in infants appears essentially normal but is somewhat dilated and tortuous during its course, especially the proximal portions. In those surviving to adult life, it is grossly dilated and tortuous, assuming aneurysmal proportions at times. Further, in adults with this condition who have reached the third or fourth decade of life, the right coronary shows hypertrophy and frequently atheromatous deposits. In contrast the left coronary is smaller in calibre and is more apt to be thinner-walled and may resemble a venous channel.

The left coronary arises from the pulmonary artery either behind the left cusp of the pulmonary valve or the posterior cusp. The distribution between these two sites is approximately equal. The usual branches arise from the left coronary and their course is that found in a normal subject.

The endocardium of the left ventricle invariably shows some degree of endocardial fibroelastosis (Fig. 2). This may be greater beneath the area of the left ventricle involved with patchy fibrosis. It is more diffuse and thinner histologically than that commonly seen in babies with primary endocardial fibroelastosis and normally placed coronary arteries. It seems possible that the interference with the circulation to the left heart may damage the structure of the endocardium sufficiently to encourage the deposition of fibrin.

Still and Boult (1956) have presented evidence that the lesion in primary endocardial fibroelastosis is associated with fine layers of fibrin on the endocardium, and such may well be the case in the lesion under discussion here.

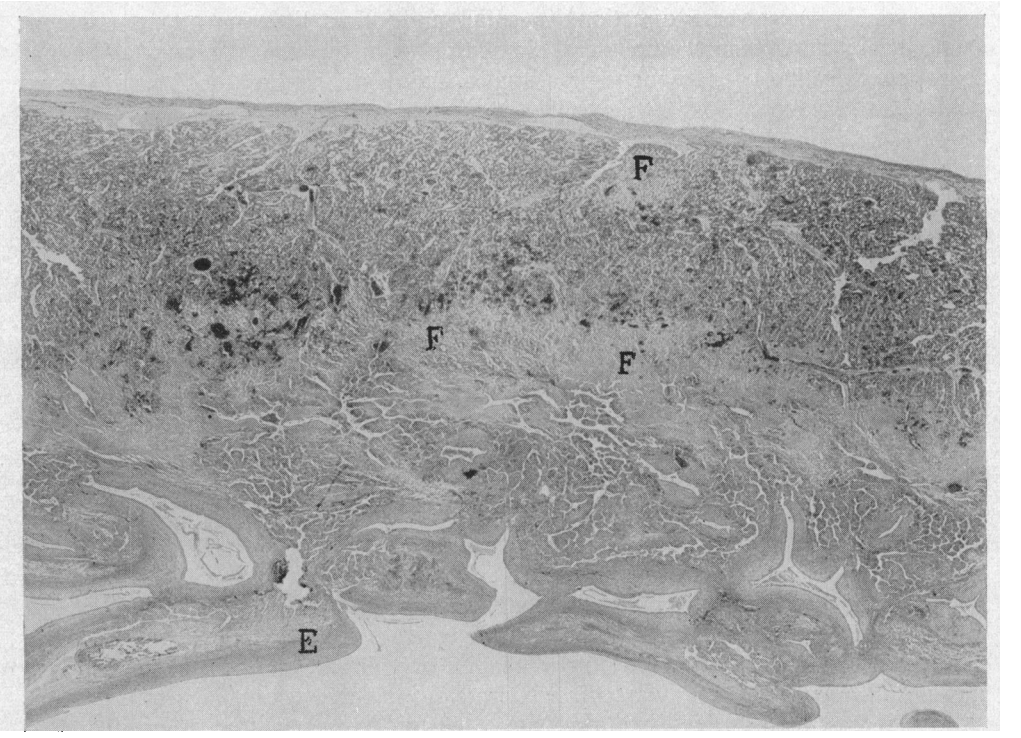

FIG. 2.—D.C., died at 5 months age. Section of left ventricle (anterior), showing patchy fibrosis of myocardium (F). Approximately one-third of the muscle mass has been replaced with fibrotic tissue. The endocardial tissues (E) show reaction similar to that seen in idiopathic endocardial fibro-elastosis.

The incidence is nearly equally divided between the sexes, being slightly more common in female than in male patients (Kaunitz, 1947).

The lungs are frequently involved with congestive heart failure, and may show patchy pneumonia and focal atelectasis.

Hæmodynamics. If the pressure in the pulmonary artery is high enough to overcome the arterial and capillary resistance of the left coronary artery, the direction of flow will be in the usual manner delivering blood into the coronary sinus. At best, however, such a coronary artery arising from the pulmonary will be receiving venous blood under a relatively low pressure. If, however, the collateral circulation from the right coronary is sufficiently large the direction of flow may be reversed and the anomalous left coronary then acts as a vein, conveying the blood into the pulmonary artery. At birth, when the pressure in the right ventricle and pulmonary artery is at or near normal systemic level, the flow will be in the normal direction. As the pressure falls in the right ventricle and pulmonary artery in a matter of hours or days after birth, the flow will diminish until it approximates to that coming from the right coronary via the collaterals. In infants with this anomaly who have heart failure the pressure may become raised again in the pulmonary artery to a degree that will permit a flow through the anomalous coronary in the usual direction. Bland and White speculated on this mechanism in 1933.

Only one of 5 cases, in which angiocardiographic studies have been carried out, revealed opacification of the aberrant left coronary. In the other 4, the contrast medium filled the pulmonary artery vividly but revealed no evidence of the anomalous left coronary in spite of the fact that its presence was subsequently confirmed at autopsy or operation. Thus, both clinical and pathological evidence suggests that the flow through the aberrant left coronary artery, whether in the

conventional or in the reverse direction, is small and offers little or no nourishment to the tissues supplied by it. When the collateral circulation from the right coronary at the margins of the areas supplied by the left is poor, severe myocardial degeneration and fibrosis will occur and lead to early death. When it is large, survival is possible into adult life.

Clinical Features

Infants with this anomaly appear normal at birth and continue to present a healthy appearance for the first month as a rule. Apparently normal development may continue until the second half of the first year, but the majority do not survive the first six months, and have usually had signs or symptoms for several weeks preceding the terminal event. In many the interval is short, often less than a week, and one died after a severe crying spell, without any previous evidence of the disease (Swann, 1955). Approximately one-third have intermittent or persistent evidence of illness of four to six weeks duration. A smaller proportion have been a problem from two to four months before death.

The evidence of illness may be grouped under three headings: (1) discomfort, (2) heart failure, and (3) respiratory infections.

In many instances there are no signs and symptoms until the onset of congestive heart failure. In a fair proportion of babies with this anomaly there is a history of irritability and discomfort for several weeks or days previously. Distress has been reported after feeding and has been sufficiently severe occasionally to suggest anginal origin. Paroxysms of distress with pallor, sweating, and dyspnœa have also been noted, precipitated or made worse by feedings.

In the cases previously reported upper respiratory disease has been a common event, occurring intermittently and usually associated with signs in the chest or bronchitis or pneumonia. Such evidence is less likely to lead one away from the underlying cardiac origin of the condition since an X-ray or clinical and radiological examination of the heart will reveal an enlarged cardiac shadow and initiate further investigation.

The symptoms of chief importance, however, are intimately associated with the onset of heart failure, and they include: dyspnœa, tachycardia, wheezing respirations, cough, and occasionally secondary cyanosis. Dyspnœa and increase in respiratory rate occur in all cases at some time during their illness.

The Electrocardiogram. The electrocardiogram in anomalous origin of the left coronary from the pulmonary artery is usually characteristic. The findings in 22 cases are summarized below. They include 10 of our own cases and 13 from reports of other pædiatric centres (Fig. 3, 4, 5 and 6).

The rate varied from 110–260; the conduction time from 0·09–0·14 sec.; the QRS time 0·09–0·10 sec.; and the axis deviation from 60° to 90°. The electrical position was usually horizontal.

Standard leads: T_1 inverted (22 of 23 cases), T_2 inverted (10 of 23 cases), T_3 inverted (2of 23 cases).

S–T segment lead 1: Slightly raised (10 of 23 cases), slightly depressed (8 of 23 cases).

qR pattern in lead 1: Occurred in 20 of 23 cases. In 2 of the remaining 3 the qR portion of the curve was slurred.

qR pattern in unipolar limb lead AVL: A qr pattern was present in all cases in which AVL was recorded (13 cases). The Q wave was almost invariably more than 50 per cent of the R wave, but in one case it was 20 per cent. The T wave was inverted in AVL in 12 of 14 cases; flat in 1, and upright in 1.

Præcordial leads (18 cases): Evidence of left ventricular hypertrophy present in all cases (usually indicated by a low ratio in R/S (less than 1) in V1.

T waves: Inverted T in V5 or V6 in 12 of 18 cases (V4 was inverted in only one case).

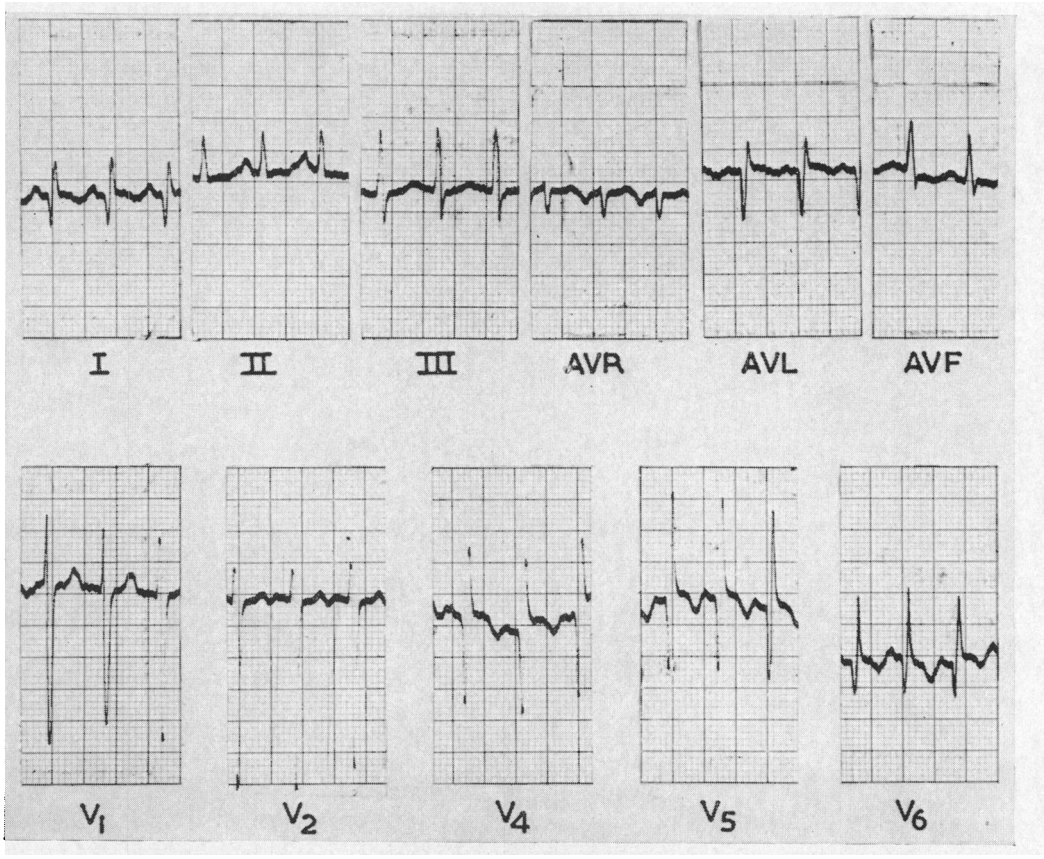

FIG. 3.—I.K. Dyspnœa from 5 weeks of age. Died at 7 weeks of age. Electrocardiogram taken the day before death. Necropsy control.

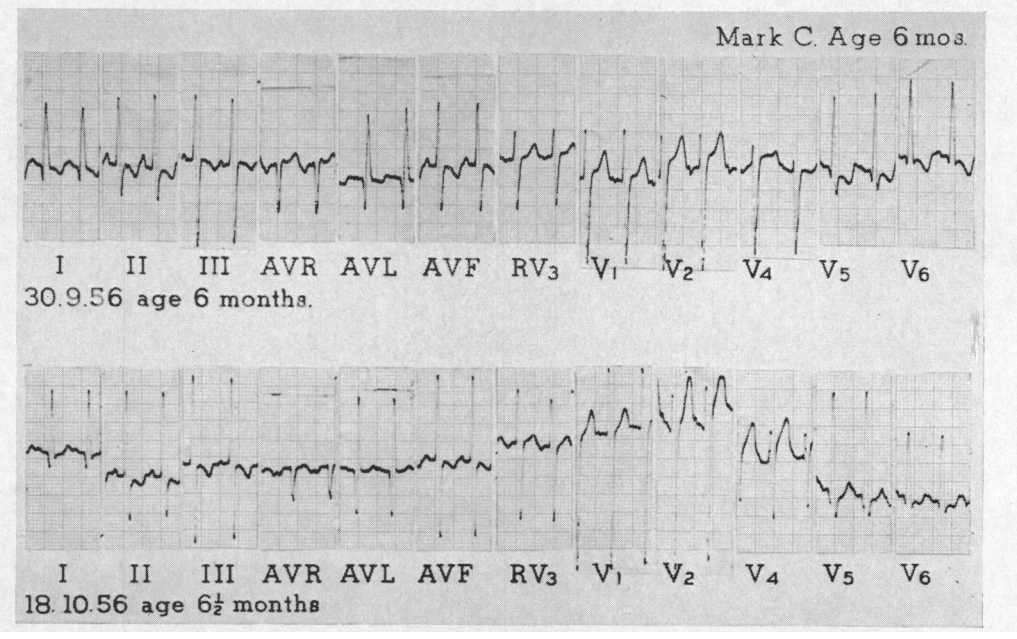

FIG. 4.—M.C. Dyspnœa for one week before the electrocardiograms were taken at 6 and 6½ months of age. He died four days later after operation.

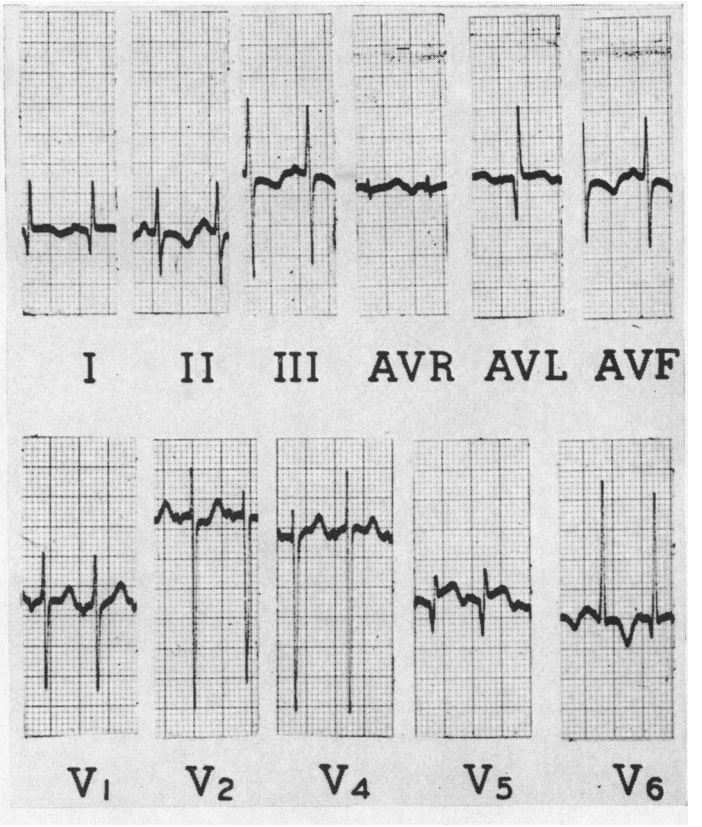

FIG. 5.—D.C. Wheezing respiration from 3 months, electrocardiogram at three weeks. He died at 5½ months. Necropsy control.

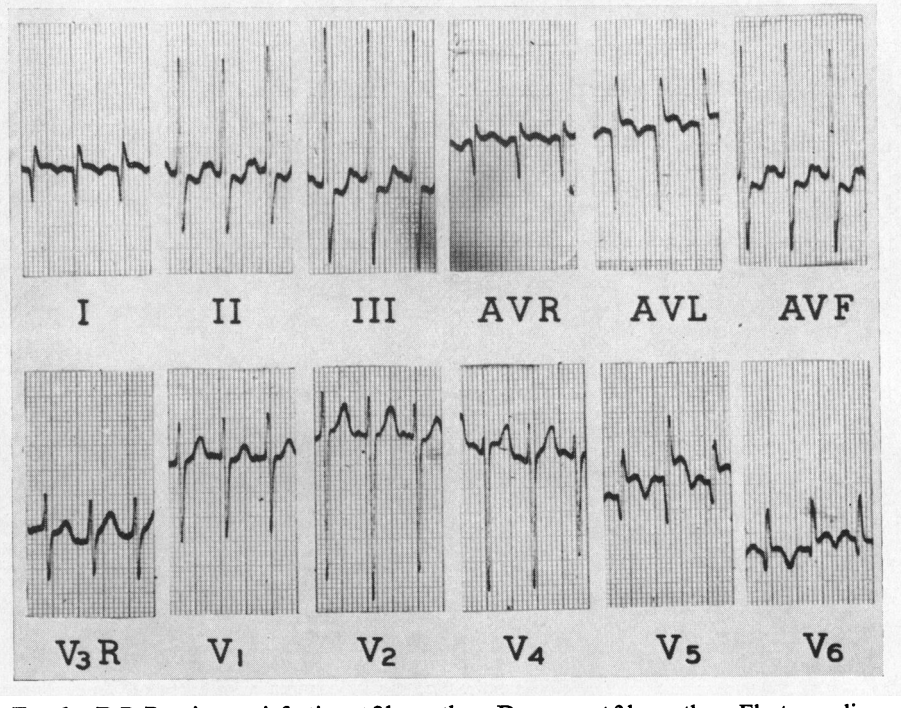

FIG. 6.—D.P. Respiratory infection at 2½ months. Dyspnœa at 3½ months. Electrocardiogram one week before death. Necropsy control.

S–T Segment: Raised S–T segment over the left præcordium in 11 of 18 cases. Usually evident in V5. S–T depressed slightly in 4 of 18 cases. No deviation of S–T in 3 of 16 cases. S–T segment usually cove-shaped when elevated.

Q waves in left præcordium: Deep Q waves usually present in V5 and V6.

The electrocardiographic data show a rapid rate, a relatively short conduction time, an axis that is usually +30° or 0°, but may vary between −60° and +90°. The electrical position is characteristically horizontal, which is an uncommon finding in this age group in the non-cyanotic heart anomalies.

The T wave is almost invariably inverted in standard lead 1, and is associated with a qR pattern in the same lead in all but a few exceptional cases. This was first pointed out by Vlad (1955) (Fig. 2). The exceptional cases that do not have a qR pattern usually show a slurring of the qR portion, an abnormality in AVL with a Q wave approximately half the R wave.

The præcordial leads commonly show inverted T waves in V5 or V6, and either slight elevation or slight depression of the S–T segment over the left præcordium. There is evidence of left ventricular hypertrophy in all cases, indicated by the low R/S ratio in V1.

As is evident, these findings are characteristic of anterior myocardial infarction in adults. In the infants, however, there is no obstruction to the descending branch of the left coronary, but the area supplied by it shows a patchy cellular necrosis and fibrosis which is undoubtedly responsible for the electrocardiographic patterns described above. This complete pattern was first pointed out by Holzmann (1951).

The tracing may change over a few days or weeks, sometimes with evidence of improvement and a return of the electrocardiogram closer to normal patterns. This is especially true of the cases that respond temporarily to digitalis.

Radiology. The radiographic appearance of the heart in 6 cases is shown in Fig. 7. Enlargement of the heart is always present, with the apex protruding down and out into the left axilla. The left cardiac border is full and convex, the right atrial shadow is full and enlarged, particularly when failure is present. The lung fields may be congested. In the left anterior oblique position the chief feature is an enlargement of the left ventricle which protrudes back overlapping the vertebral column to a marked degree.

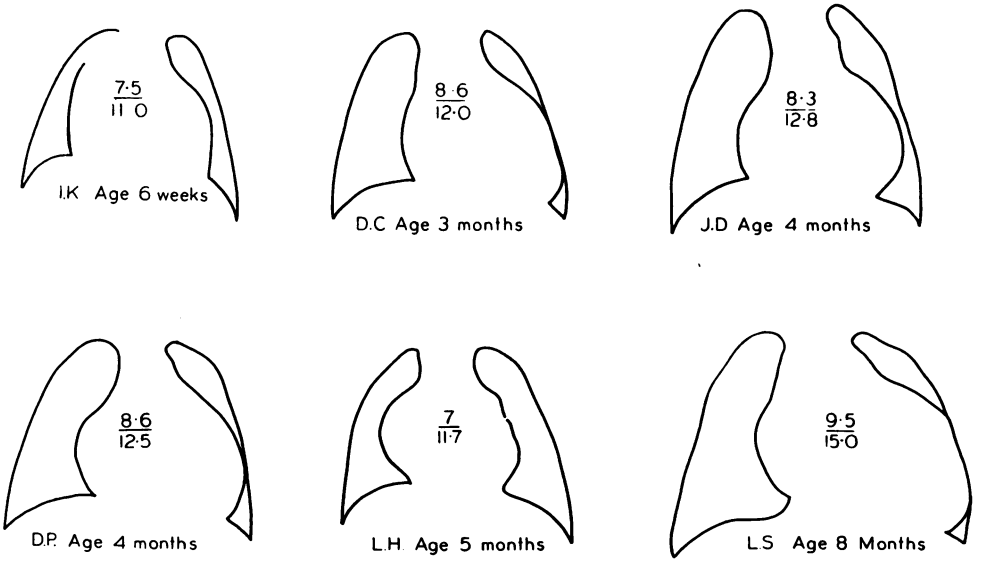

FIG. 7.—Anomalous origin of the left coronary from the pulmonary artery. Outlines of six cardiac shadows of infants with anomalous left coronary arteries. Enlargement with convex cardiac borders demonstrated.

Under the fluoroscope the cardiac borders appear quiet, while the left ventricle is enlarged in all views. A barium swallow demonstrates a normal left aortic arch. The congested hilar shadows show no evidence of pulsation. The left atrium is frequently within normal limits in size but may be slightly or moderately enlarged. Such enlargement is probably due to the enlarged left ventricle pushing the atrium back, plus the presence of increased pressure from the failing left ventricle. The right ventricle shows no enlargement but its border in the left anterior oblique view may appear more active than that of the left ventricle.

Angiocardiography. Angiocardiography provides useful information in making a definitive diagnosis. A venous angiogram will demonstrate a dramatically small right ventricle compressed into a diminutive portion of the cardiac shadow, and giving rise to a normal pulmonary artery and its branches (Fig. 8). The left ventricle is greatly enlarged, rounded, and occupies nearly two-thirds of the cardiac outline. Its emptying is delayed. By examining the angiogram of the left ventricle, one can determine the thickness of its wall. This is of some importance since dilatation is more a feature than hypertrophy in this condition. (The reverse is true in endocardial fibroelastosis).

Visualization of the left coronary artery arising from the pulmonary artery sinuses has been noted once (Taussig, 1956), but demonstration of the anomalous coronary by this method is the exception rather than the rule because of the lower pressure in the pulmonary circulation, and the

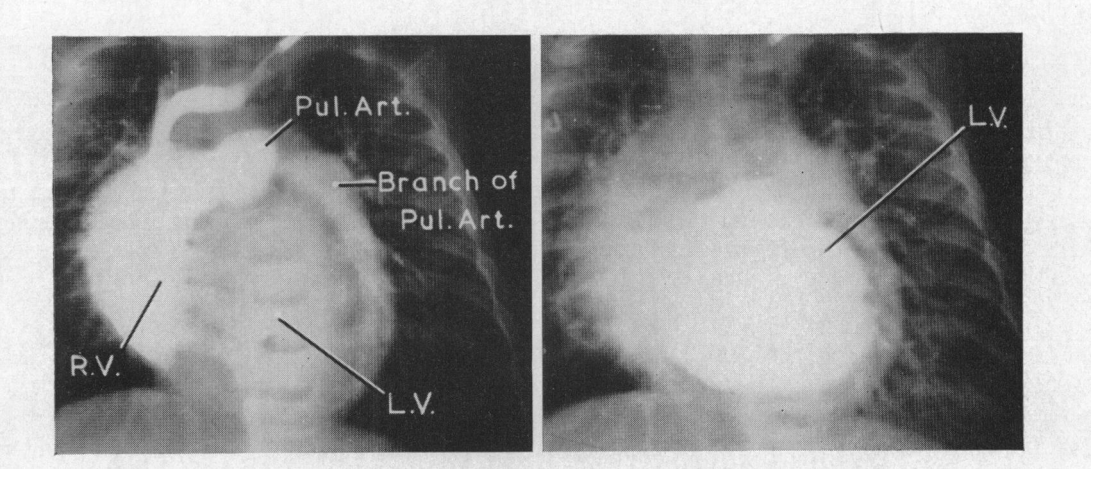

FIG. 8.—L.H. 15 months 24 days. Venous angiogram of baby with auricular left coronary, aged approximately 16 months. Small right ventricle and large left ventricle clearly shown. No evidence of left coronary filling from the pulmonary artery.

higher resistance in the coronary vessels. Out of five venous or selective angiograms seen by the author, only the one referred to above showed any evidence of the left coronary filling from the pulmonary artery.

A more profitable procedure is to do a selective aortogram with the tip of the catheter inserted into the femoral artery and slid up into position immediately above the aortic valves. An injection of a few ml. of contrast medium will fill the coronary arteries in a normal infant. When the left coronary arises from the pulmonary artery only the right coronary will be visualized emerging from the aorta (Fig. 9). It is also of interest that the right coronary will then frequently appear dilated and tortuous since besides its own circulation it is providing blood to part of the left ventricle via collaterals.

Cardiac Catheterization. We have performed cardiac catheterization on three infants with the left coronary arising from the pulmonary artery. None of these presented clear-cut signs of failure

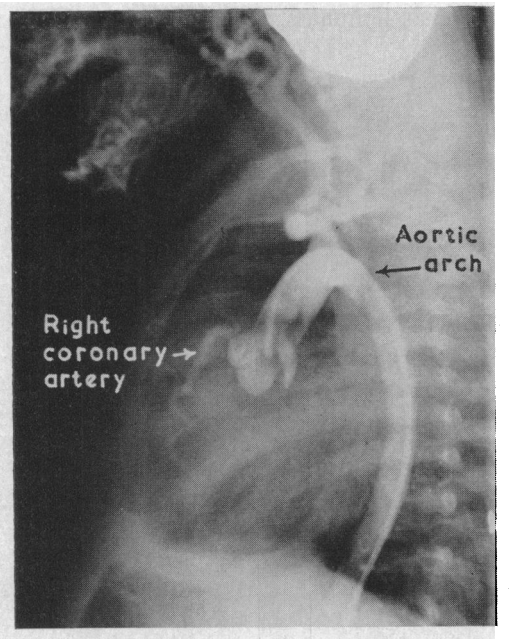

FIG. 9.—M.C. Selective aortogram with tip of the catheter just above the aortic valve. Right coronary artery is shown filling clearly but no evidence of left coronary. At necropsy it was demonstrated to be coming off the pulmonary artery.

at the time of catheter study, although all had previously, before being digitalized. These findings were as follows.

		IVC	SVC	RA	RV	PA	PA wedge	Br. artery
L.S.	8½ mo.	49%	41%	35% 6/−2	37% 30/0	42% 26/11	mean 12	110/60 (cuff)
M.C.	6 mo.		54%	48%	54% 38/2	55% 28/15	11	120/50 (cuff)
L.H.	2½ mo.	72%	58%	66%	69% 55/15	67% 50/20	10	80/40 (cuff)

These data show a wide variation in oxygen saturation of the venous blood. The highest pressure in the right ventricle and pulmonary artery was not accompanied by the lowest degree of venous oxygen saturation. The pulmonary capillary pressure was significantly increased in two cases where it was obtained. In each case the pressure in the pulmonary artery was above average but in only one was it significantly above the upper limit of normal, and that was in the youngest case (age 2½ months). One case had a regurgitant jet through the mitral valve when explored surgically.

DIAGNOSIS

A diagnosis may now be made during life in those cases that present with signs and symptoms in the pædiatric age group. It may be based on the following criteria.

The baby will be in the first year of life almost invariably. The majority are brought to the

physician between the second and sixth months. Signs of congestive heart failure may be apparent, with dyspnœa or wheezy respirations. There may be a history of distress during or after feeding. Previous attacks of respiratory infections or pneumonia are common.

Physical examination shows a baby that is irritable and lacks normal energy. Breathing may be normal or rapid. The lips are of good colour except in those cases of severe congestive failure, when minimal cyanosis may be recognized. X-ray of the chest shows a moderate to grossly enlarged heart, generalized fullness of the left cardiac border, and an especially large left ventricle. The electrocardiogram is most useful since it usually reveals a diagnostic pattern. Such a tracing is consistent with anterior myocardial fibrosis, and is characterized by a qR pattern, an inverted T in standard lead 1, and a similar configuration in aVL. This is coupled with evidence of left ventricular hypertrophy in the præcordial leads, a deep Q wave in V5 and V6 and usually an inversion of the T waves over the left præcordium, often with a cove-shaped S–T. These findings in a baby with congestive heart failure in the first year of life, are highly suggestive of an anomalous left coronary. To confirm the diagnosis a selective aortogram with the tip of the catheter in the aorta just above the aortic valve will show filling of the right coronary artery, and no filling of the left, thus indirectly establishing the diagnosis.

A venous angiogram, while not likely to demonstrate the anomalous coronary may show up a large, relatively thin-walled left ventricle, and a small compressed right ventricle.

The differential diagnosis includes those congenital heart anomalies that are associated with left ventricular hypertrophy or enlargement, such as endocardial fibroelastosis, tricuspid atresia, aortic stenosis, patent ductus arteriosus, coarctation of the aorta, ventricular septal defect, myocarditis, and coronary calcinosis. Most of these can be differentiated with relative ease. The problem that presents the greatest difficulty is to differentiate between endocardial fibroelastosis and an aberrant left coronary arising from the pulmonary. Since both are likely to be found in the first year of life, age is not of much value in differentiation. However, when the onset of symptoms occurs in the second year of life, one is more likely dealing with endocardial fibroelastosis than aberrant left coronary. The electrocardiogram will usually permit a definitive diagnosis since the aberrant left coronary gives a pattern of anterior myocardial fibrosis associated with left ventricular hypertrophy, while endocardial fibroelastosis simply presents evidence of left ventricular hypertrophy (Vlad *et al.*, 1955). An elevation of the S–T segment in the left præcordial leads is rare in endocardial fibroelastosis and common in the aberrant coronary. T wave inversion over the left præcordial leads frequently involves all the complexes in the left præcordium in endocardial fibroelastosis, whereas it is usually found in V5 or V6 in the aberrant coronary cases. A large Q wave followed by an inverted T wave is present almost invariably in aVL in the aberrant coronary cases, whereas this pattern is rarely found in endocardial fibroelastosis.

Tricuspid atresia is not likely to constitute a problem since it is associated with obvious cyanosis. The murmur usually differentiates aortic stenosis, although on rare occasions in early life aortic stenosis may show a deeply inverted T wave over the left præcordium associated with heart failure, and no murmur. However, the infarct or fibrotic pattern is lacking. Patent ductus arteriosus and ventricular septal defect can be readily differentiated. Comparison of blood pressures in arm and leg will rule out coarctation of the aorta. Myocarditis may present inverted T waves over the left præcordium, but Q wave and S–T changes of anterior myocardial fibrosis referred to above are lacking. Coronary calcinosis may lead to left ventricular hypertrophy. However, an ischæmic pattern may be present in the electrocardiogram. A vertical or semivertical position is characteristic of coronary calcinosis and is therefore like endocardial fibroelastosis. A horizontal or an intermediate position is usually found in left coronary from the pulmonary artery.

Prognosis

From the evidence at hand the prognosis is poor. There are two obvious groups divided by age. The first and largest, comprising approximately 85 per cent of the total, is the group of infants presenting with signs or symptoms in the first year of life. Such babies do not survive for long.

The age at death is shown in the accompanying chart (Fig. 10). It will be noted that all were dead by the end of the second year of life. The largest number die in the first six months usually in the third or fourth month. These babies die of congestive heart failure in most instances, but a few die a sudden death with minimal evidence of failure. A respiratory infection may precipitate the final event.

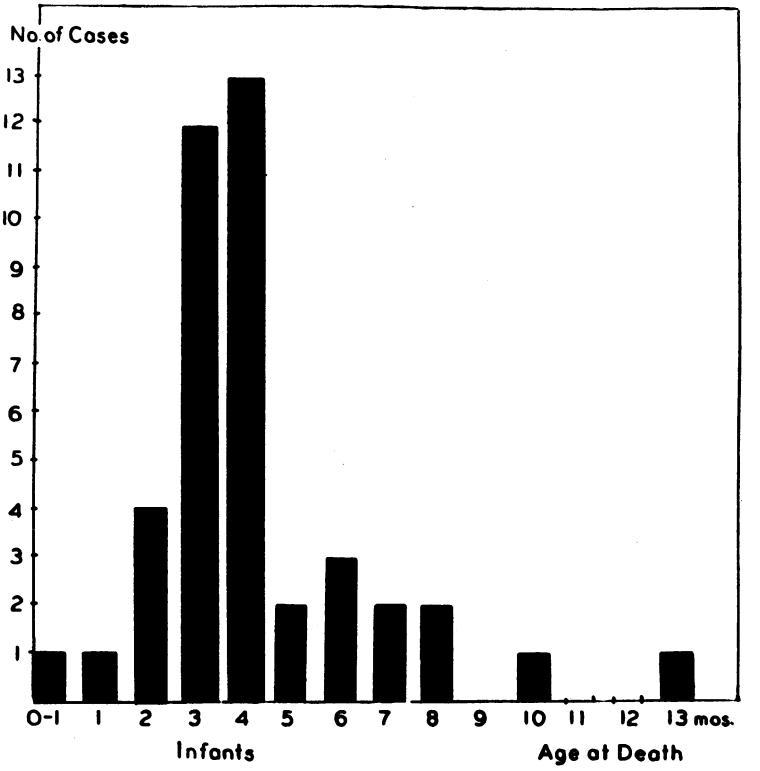

FIG. 10.—Anomalous origin of the left coronary from the pulmonary artery. Age at death in infant group. Most of these infants die in first 3 or 4 months of life. A few survive the first year of life with continued signs of heart failure. Those who have been discovered in adult life had no signs or symptoms in infancy.

The second group (not shown in figure), comprising approximately 15 per cent of the total, survive to adult life and have an average age at death of thirty to forty years. One patient survived to sixty-four years (Abbott). These cases have not been recognized during infancy or childhood and most had led relatively active lives. In this group sudden and unexpected death may occur, or it may be ushered in by congestive heart failure. Several instances had a history of angina attacks extending over a period of several years.

The first group die in infancy because of an inadequate circulation to the left ventricle. The second group die in the third or fourth decade for the same reason. The difference between the two groups is a matter of the degree of collateral circulation. While the latter group survive to adult life their prognosis is far short of the normal span by modern standards.

TREATMENT

Surgical. A variety of suggestions have been made regarding surgical therapy in this condition. These include the following.

(1) The creation of an artificial ductus by Blalock type of anastomosis that would serve the purpose of increasing the oxygen content of the blood in the main pulmonary artery.

(2) Producing a constriction of the pulmonary artery just beyond the origin of the left coronary in order to raise the pressure in the right ventricle and thus the aberrant coronary.

(3) The introduction of talcum powder into the pericardial cavity to increase the collateral circulation to the left ventricle. This was done in a case described by Paul and Robbins (1955). The baby survived the operation and appeared to be somewhat improved by it.

(4) The technique of Vineberg and Buller (1955) of inserting the internal mammary artery into the left ventricle. The vessels in infants are very small and it is doubtful whether these infants would survive the procedure.

(5) The anastomosis of a systemic artery to the left coronary, after the latter had been removed from its aberrant origin from the pulmonary artery. This has been attempted by Mustard (1954). Technically the procedure was completed, but the infant died immediately following the operation.

(6) Currently infants have been treated by ligation of the left coronary artery in an effort to prevent that vessel from acting as a vein and draining off collateral flow into the pulmonary artery. Optimally this may have the effect of increasing the collateral flow from the right coronary into the undernourished left ventricular tissue. Mustard (1956) has tried this once. The baby survived for eight hours post-operatively. Jenke (1957) recently reported a successful case in an infant who survived and has been improved by it.

(7) Apley *et al.* (1957) have recently suggested that Murray's technique of dissecting out myocardial infarctions be adapted to this condition. They have been brought to this point of view by a baby that died under their care following attempted surgery at nine months of age, and at necropsy injection of the left coronary revealed obliteration of many of its branches. Thus anastomosis, it was suggested, would have been of little benefit. This may well be true, but, with an adequate blood supply at its origin, the left coronary branches might well be patent in the majority of cases recognized before the terminal stages were reached. Even in the more advanced cases new channels might conceivably open up, especially in the younger patients. Regeneration of functioning myocardium may occur because the involvement is patchy —not uniform (see Fig. 2).

Ligation of the anomalous left coronary at its origin still leaves left ventricular muscles with inadequate flow and does not preclude the possibility of sudden death or heart failure during childhood or early adult life. The operation of choice would appear to be an anastomosis between a systemic artery and the anomalous left coronary, especially if done early in the course. Mustard has demonstrated that this is mechanically feasible. Modern techniques may make it a practical achievement in the future.

Medical. Temporary improvement can be achieved in most cases that present with congestive heart failure provided they are not in the final stages of the syndrome. A response to digitalis is common, leading to a diminution in liver size and decrease in respiratory rate and the clearing of chest signs. The electrocardiogram may also show improvement with the T waves in the left præcordium reverting to an upright position or a return of the S–T segment to the base line. However, all these signs of regression are temporary, and in the course of a few weeks there is a return of congestive heart failure leading to a fatal outcome.

The importance of this transient improvement, however, is that it may give the surgeon a chance to carry out a procedure with greater opportunity of success.

Summary

The anomalous origin of the left coronary artery from the pulmonary artery is a rare defect occurring once in 300,000 children. It usually causes death within the first year of life and at necropsy is characterized by an enlarged, somewhat thin-walled, left ventricle, and a patchy fibrosis in the part of the left ventricle supplied by the anomalous coronary. The pathological change would appear to be related to the inadequate blood supply, more than to the oxygen content of the blood delivered.

The diagnosis should be suspected in an infant in the first year of life who develops signs of heart failure, an enlarged heart, and an electrocardiogram with evidence of myocardial fibrosis in the

anterior portion of the left ventricle. The diagnosis may be largely confirmed by an aortogram carried out with the catheter just above the aortic valve. This will reveal filling of the right coronary from the aorta but will not reveal any filling of the left coronary.

Adequate surgery has not yet been evolved but it would appear possible that an anastomosis might be completed between a segment of the aorta at the site of the left coronary and a systemic artery. If this was carried out early enough before advanced myocardial change had occurred normal heart function might result.

I am indebted to MacMillan Co., New York, for permission to produce Fig. 9 from *Heart Disease in Infancy and Childhood*, by Keith *et al.*

REFERENCES

Abrikosoff, A. (1911). *Arch. path. Anat.*, **203**, 413.
Apley, J., Horton, R. E., and Wilson, M. C. (1957). *Thorax*, **12**, 28.
Bassis, M. L., and Sheinkopf, J. A. (1955). *Ann. intern. Med.*, **42**, 893.
Bland, E. F., White, P. D., and Garland, J. (1933). *Amer. Heart J.*, **8**, 787.
Cronk, E. S., Sinclair, J. G., and Rigdon, R. H. (1951). *Amer. Heart J.*, **42**, 906.
Dagonet, Y. (1952). *Arch. Mal. Cœur*, **45**, 7.
Denko, J. V., and Hagerty, C. S. (1953). *A.M.A. Arch. Path.*, **56**, 142.
Eidlow, S., and Mackenzie, E. R. (1946). *Amer. Hert J.*, **32**, 243.
Gasul, B. M., and Loeffler, E. (1949). *Pediatrics*, **4**, 498.
Gouley, B. A. (1950). *Amer. Heart J.*, **40**, 630.
Hamilton, W. E. (Montreal), and Abbott, M. E. (1928). *Amer. Heart J.*, **3**, 381.
Hartenstein, H., and Freeman, D. J. (1952). *Amer. J. Dis. Child.*, **83**, 774.
Holsmann, M. (1952). *Clinical Electrocardiography.* (Translated by Robertson, D.) Staples, Press, London.
Jenke, A. (1957). Personal communication.
Kaunitz, P. E. (1947). *Amer. Heart J.*, **33**, 182.
Keizer, D. P. R., and Rochat, R. R. (1952). *Amer. J. Dis. Child.*, **83**, 769.
Kittle, C. F., Diehl, A. M., and Heilbrunn, A. (1955). *Pediatrics*, **47**, 198.
Lyon, R. A. (Cincinnati), Johansmann, R. J., and Dodd, K. (1946). *Amer. J. Dis. Child.*, **72**, 675.
Miller, R. A. (1954). *Ped. Cl. No. Amer.*, **1**, 51.
Mustard, W. T. (1956). Personal communication.
McKinley, H. I., Andrew, J., and Neill, C. A. (1951). *Pediatrics*, **8**, 828.
Paul, R. N., and Robbins, S. G. (1955). *Pediatrics*, **16**, 147.
Schlicter, J., Hellerstein, H. K., and Katz, L. N. (1954). *Medicine*, **33**, 43.
Still, W. J. S., and Boult, E. H. (1956). *Lancet*, **2**, 117.
Taussig, H. B. (1947). *Congenital Malformations of the Heart.* The Commonwealth Fund, New York, p. 320.
Tedeschi, C. G., and Helpern, M. M. (1954). *Pediatrics*, **14**, 53.
Tellem, M., and Rubenstone, A. I. (1955). *Amer. J. Med.*, **19**, 65.
Vineberg, A., and Buller, W. (1955). *J. Thorac. Surg.*, **30**, 4112.
Vlad, P. (1955). Personal communication.
——, Rowe, R. D., and Keith, J. D. (1955). *Brit. Heart J.*, **17**, 189.